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All: 208 Review: 202

Items 101 - 120 of 208 Previous Page 6 of 11 Next

101: Mannello F, Gazzanelli G. Related Articles, Links

Tissue inhibitors of metalloproteinases and programmed cell death: conundrums, controversies and potential implications.
Apoptosis. 2001 Dec;6(6):479-82. Review.
PMID: 11595838 [PubMed - indexed for MEDLINE]

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Semin Liver Dis. 2001 Aug;21(3):373-84. Review.
PMID: 11586466 [PubMed - indexed for MEDLINE]

103: Lijnen HR. Related Articles, Links

Plasmin and matrix metalloproteinases in vascular remodeling.
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PMID: 11487021 [PubMed - indexed for MEDLINE]

104: Creemers EE, Cleutjens JP, Smits JF, Daemen MJ. Related Articles, Links

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105: Nguyen M, Arkell J, Jackson CJ. Related Articles, Links

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Postepy Hig Med Dosw. 2001;55(2):303-18. Review. Polish.

PMID: 11468976 [PubMed - indexed for MEDLINE]

☐ **107:** Okazaki I, Watanabe T, Hozawa S, Niioka M, Arai M, Maruyama K. Related Articles, Links

Reversibility of hepatic fibrosis: from the first report of collagenase in the liver to the possibility of gene therapy for recovery.
Keio J Med. 2001 Jun;50(2):58-65. Review.
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Gelatinase B functions as regulator and effector in leukocyte biology.
J Leukoc Biol. 2001 Jun;69(6):851-9. Review.
PMID: 11404367 [PubMed - indexed for MEDLINE]

☐ **109:** Licht P, Russu V, Wildt L. Related Articles, Links

On the role of human chorionic gonadotropin (hCG) in the embryo-endometrial microenvironment: implications for differentiation and implantation.
Semin Reprod Med. 2001;19(1):37-47. Review.
PMID: 11394202 [PubMed - indexed for MEDLINE]

☐ **110:** Elliott S, Cawston T. Related Articles, Links

The clinical potential of matrix metalloproteinase inhibitors in the rheumatic disorders.
Drugs Aging. 2001;18(2):87-99. Review.
PMID: 11346130 [PubMed - indexed for MEDLINE]

☐ **111:** Kossakowska AE, Urbanski SJ, Janowska-Wieczorek A. Related Articles, Links

Matrix metalloproteinases and their tissue inhibitors - expression, role and regulation in human malignant non-Hodgkin's lymphomas.
Leuk Lymphoma. 2000 Nov;39(5-6):485-93. Review.
PMID: 11342332 [PubMed - indexed for MEDLINE]

☐ **112:** Curry TE Jr, Osteen KG. Related Articles, Links

Cyclic changes in the matrix metalloproteinase system in the ovary and uterus.
Biol Reprod. 2001 May;64(5):1285-96. Review.
PMID: 11319131 [PubMed - indexed for MEDLINE]

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A putative role for oncogenes in trophoblast invasion?
Hum Reprod. 2000 Dec;15 Suppl 6:51-8. Review.
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☐ **116:** Johnson PR. Related Articles, Links

Role of human airway smooth muscle in altered extracellular matrix production in asthma.
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Methods Mol Biol. 2001;151:121-48. Review. No abstract available.
PMID: 11217296 [PubMed - indexed for MEDLINE]

☐ **118:** Kraiem Z, Korem S. Related Articles, Links

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Thyroid. 2000 Dec;10(12):1061-9. Review.
PMID: 11201850 [PubMed - indexed for MEDLINE]

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Semin Cancer Biol. 2000 Dec;10(6):415-33. Review.
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PMID: 11147675 [PubMed - indexed for MEDLINE]

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L2	364	mmp near4 inhibition	USPAT	OR	OFF	2005/11/21 12:52
L3	29	timp near4 inhibition	USPAT	OR	OFF	2005/11/21 12:52
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L4           158 L1 AND L2 AND L3

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L6            3 L5 AND REVIEW

=> d l5 30-62 bib

L5   ANSWER 30 OF 62     MEDLINE on STN                    DUPLICATE 24
AN   2002741786     MEDLINE
DN   PubMed ID: 12505230
TI   Release of matrix metalloproteinases following alcohol septal
ablation in
     hypertrophic obstructive cardiomyopathy.
AU   Bradham William S; Gunasinghe Himali; Holder Jennifer R; Multani
Marlina;
     Killip Donna; Anderson Marianne; Meyer Denise; Spencer William H
3rd;
     Torre-Amione Guillermo; Spinale Francis G
CS   Medical University of South Carolina, Charleston 29425, USA.
NC   HL59165 (NHLBI)
     M01 RR01070 (NCRR)
     P01 HL48788-08 (NHLBI)
SO   Journal of the American College of Cardiology, (2002 Dec 18) 40
(12)
     2165-73.
     Journal code: 8301365. ISSN: 0735-1097.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Abridged Index Medicus Journals; Priority Journals
EM   200302
ED   Entered STN: 20021231
     Last Updated on STN: 20030215
     Entered Medline: 20030214

L5   ANSWER 31 OF 62     MEDLINE on STN                    DUPLICATE 25
AN   2002219946     MEDLINE
DN   PubMed ID: 11956636
TI   Recent advances in the regulation of matrix metalloproteinase 2
     activation: from basic research to clinical implication
(Review).
AU   Yoshizaki Tomokazu; Sato Hiroshi; Furukawa Mitsuru
CS   Department of Otolaryngology, Graduate School of Medicine,
Kanazawa
     920-8641, Japan.. tomoy@orl.m.kanazawa-u.ac.jp
SO   Oncology reports, (2002 May-Jun) 9 (3) 607-11.  Ref: 57
     Journal code: 9422756. ISSN: 1021-335X.
```

CY Greece
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200210
ED Entered STN: 20020417
Last Updated on STN: 20021010
Entered Medline: 20021008

L5 ANSWER 32 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:88099 CAPLUS
DN 138:399731
TI The inhibitory effect of TIMP-1 on the spontaneous liver fibrosis resolution in a transgenic mouse model
AU Yoshiji, Hitoshi; Yoshii, Junichi; Ikenaka, Yasuhide; Noguchi, Ryuichi;
Yanase, Koji; Namisaki, Tadashi; Fukui, Hiroshi
CS Third Department of Internal Medicine, Nara Medical University, Japan
SO Japanese Pharmacology & Therapeutics (2002), 30(Suppl. 2), S343-S348
CODEN: JPTABU
PB Raifu Saiensu Shuppan K.K.
DT Journal
LA Japanese

L5 ANSWER 33 OF 62 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
STN
AN 2003:155230 BIOSIS
DN PREV200300155230
TI Degradation Of Timp-1 And Increased Gelatinase Activity In Human Stromal
Cell Cultures Treated With Nitric Oxide By-products.
AU Kenney, M. C. [Reprint Author]; Lin, B. [Reprint Author]; Chwa, M.
[Reprint Author]; Saghizadeh, M. [Reprint Author]; Rosenberg, S. [Reprint
Author]; Ljubimov, A. V. [Reprint Author]; Brown, D. J. [Reprint Author]
CS Ophthalmology Research, Cedars-Sinai Medical Center, Los Angeles, CA, USA
SO ARVO Annual Meeting Abstract Search and Program Planner, (2002) Vol. 2002,
pp. Abstract No. 3208. cd-rom.
Meeting Info.: Annual Meeting of the Association For Research in Vision
and Ophthalmology. Fort Lauderdale, Florida, USA. May 05-10, 2002.
DT Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 26 Mar 2003
Last Updated on STN: 26 Mar 2003

L5 ANSWER 34 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:597119 CAPLUS
DN 135:352449
TI Inhibition of Wilms' tumor growth by intramuscular administration of
tissue inhibitor of metalloproteinases-4 plasmid DNA
AU Celiker, M. Y.; Wang, M.; Atsidaftos, E.; Liu, X.; Liu, Y. E.; Jiang, Y.;
Valderrama, E.; Goldberg, I. D.; Shi, Y. E.
CS Division of Pediatric Hematology/Oncology, Schneider Children's Hospital,
Long Island Jewish Medical Center, New York, NY, 11040, USA
SO Oncogene (2001), 20(32), 4337-4343
CODEN: ONCNES; ISSN: 0950-9232
PB Nature Publishing Group
DT Journal
LA English
RE.CNT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 35 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:111082 CAPLUS
DN 134:173401
TI Tissue inhibitor of metalloproteinases-2 (TIMP-2) suppresses TKR-growth
factor signaling independent of metalloproteinase inhibition
AU Hoegy, Susan E.; Oh, Hae-Ryong; Corcoran, Marta L.; Stetler-Stevenson,
William G.
CS Extracellular Matrix Pathology Section, Laboratory of Pathology, Division
of Clinical Sciences, National Institutes of Health, Bethesda, MD,
20892-1500, USA
SO Journal of Biological Chemistry (2001), 276(5), 3203-3214
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
RE.CNT 67 THERE ARE 67 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 36 OF 62 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
STN
AN 2001:258630 BIOSIS
DN PREV200100258630

TI Fibronectin upregulates TGFbeta-1 mediated TIMP-1 gene and protein
synthesis in neonatal foreskin fibroblasts.
AU Fasehun, Funmi A. [Reprint author]; Duran, Walter N. [Reprint author];
Hobson, Robert W., II; Pappas, Peter J.
CS University of Medicine and Dentistry, 185 South Orange Avenue, Newark, NJ,
07103, USA
SO FASEB Journal, (March 8, 2001) Vol. 15, No. 5, pp. A1129. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for
Experimental Biology on Experimental Biology 2001. Orlando, Florida, USA.
March 31-April 04, 2001.
CODEN: FAJOEC. ISSN: 0892-6638.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 30 May 2001
Last Updated on STN: 19 Feb 2002

L5 ANSWER 37 OF 62 MEDLINE on STN DUPLICATE 26
AN 2001447813 MEDLINE
DN PubMed ID: 11327240
TI Modulation of TIMP-1 synthesis by antiinflammatory cytokines and
prostaglandin E2 in interleukin 17 stimulated human monocytes/macrophages.
AU Jovanovic D V; Di Battista J A; Martel-Pelletier J; Reboul P; He Y;
Jolicoeur F C; Pelletier J P
CS Osteoarthritis Research Unit, H pital Notre-Dame, Centre Hospitalier de
l'Universite de Montreal, Quebec, Canada.
SO Journal of rheumatology, (2001 Apr) 28 (4) 712-8.
Journal code: 7501984. ISSN: 0315-162X.
CY Canada
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200108
ED Entered STN: 20010813
Last Updated on STN: 20010813
Entered Medline: 20010809

L5 ANSWER 38 OF 62 MEDLINE on STN DUPLICATE 27
AN 2001179938 MEDLINE
DN PubMed ID: 11168376
TI Thioredoxin alters the matrix metalloproteinase/tissue inhibitors of
metalloproteinase balance and stimulates human SK-N-SH neuroblastoma cell

invasion.
AU Farina A R; Tacconelli A; Cappabianca L; Masciulli M P; Holmgren A;
Beckett G J; Gulino A; Mackay A R
CS Section of Molecular Pathology, Department of Experimental Medicine,
University of L'Aquila, Via Vetoio, Coppito II, 61700 L'Aquila, Italy.
SO European journal of biochemistry / FEBS, (2001 Jan) 268 (2) 405-13.
Journal code: 0107600. ISSN: 0014-2956.
CY Germany: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200103
ED Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010329

L5 ANSWER 39 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:810861 CAPLUS
DN 136:33772
TI **MMP-TIMP** interaction depends on residue 2 in TIMP-4
AU Stratmann, Bernd; Farr, Martin; Tschesche, Harald
CS Biochemistry I, University of Bielefeld, Faculty of Chemistry, Bielefeld,
D-33615, Germany
SO FEBS Letters (2001), 507(3), 285-287
CODEN: FEBLAL; ISSN: 0014-5793
PB Elsevier Science B.V.
DT Journal
LA English
RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 40 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:82611 CAPLUS
DN 135:32045
TI Inhibition of matrix metalloproteinases by over-expression of tissue
inhibitor of metalloproteinase-2 inhibits the growth of experimental
hemangiomas
AU Vergani, V.; Garofalo, A.; Bani, M. R.; Borsotti, P.; Parker, M. Pelina;
Drudis, T.; Mazzarol, G.; Viale, G.; Giavazzi, R.; Stetler-Stevenson, W.
G.; Taraboletti, G.
CS Department of Oncology, Mario Negri Institute for Pharmacological Research, Bergamo, 24125, Italy
SO International Journal of Cancer (2001), 91(2), 241-247

CODEN: IJCNAW; ISSN: 0020-7136
PB Wiley-Liss, Inc.
DT Journal
LA English
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 41 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:34620 CAPLUS
DN 134:160811
TI Localization of the death domain of tissue inhibitor of metalloproteinase-3 to the N terminus. Metalloproteinase inhibition is associated with proapoptotic activity
AU Bond, Mark; Murphy, Gillian; Bennett, Martin R.; Amour, Augustin; Knauper, Vera; Newby, Andrew C.; Baker, Andrew H.
CS Bristol Heart Institute, Bristol Royal Infirmary, University of Bristol, Bristol, BS2 8HW, UK
SO Journal of Biological Chemistry (2000), 275(52), 41358-41363
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 42 OF 62 MEDLINE on STN DUPLICATE 28
AN 2000175276 MEDLINE
DN PubMed ID: 10708863
TI Tissue inhibitors of metalloproteinases: evolution, structure and function.
AU Brew K; Dinakarpandian D; Nagase H
CS Department of Biochemistry and Molecular Biology, University of Miami School of Medicine, Miami, FL 33101, USA..
k.brew@molbio.med.miami.edu
NC AR39189 (NIAMS)
AR40994 (NIAMS)
SO Biochimica et biophysica acta, (2000 Mar 7) 1477 (1-2) 267-83.
Ref: 74
Journal code: 0217513. ISSN: 0006-3002.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200005
ED Entered STN: 20000606
Last Updated on STN: 20000606

Entered Medline: 20000524

L5 ANSWER 43 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2001:597440 CAPLUS
DN 135:355780
TI Inhibition of vascular smooth muscle cell migration by eNOS gene expression: Possible role of **MMP**-2, **MMP**-9 and **TIMP**-2 ratio
AU Gurjar, Milind V.; Sharma, Ram V.; Bhalla, Ramesh C.
CS Department of Anatomy and Cell Biology, University of Iowa College of Medicine, Iowa City, IA, 52242, USA
SO Portland Press Proceedings (2000), 16(Biology of Nitric Oxide, Part 7), 148
CODEN: POPPEF; ISSN: 0966-4068
PB Portland Press Ltd.
DT Journal
LA English
RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 44 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:486817 CAPLUS
DN 131:241239
TI Tissue inhibitor of matrix metalloproteinase-2 regulates matrix metalloproteinase-2 activation by modulation of membrane-type 1 matrix metalloproteinase activity in high and low invasive melanoma cell lines
AU Kurschat, Peter; Zigrino, Paola; Nischt, Roswitha; Breitkopf, Katja; Steurer, Pavlos; Klein, C. Eberhard; Krieg, Thomas; Mauch, Cornelia
CS Department of Dermatology, University of Cologne, Cologne, 50924, Germany
SO Journal of Biological Chemistry (1999), 274(30), 21056-21062
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
RE.CNT 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 45 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:467036 CAPLUS
DN 131:239611
TI The specificity of TIMP-2 for matrix metalloproteinases can be modified by single amino acid mutations
AU Butler, Georgina S.; Hutton, Mike; Wattam, Beth A.; Williamson, Richard

A.; Knauper, Vera; Willenbrock, Frances; Murphy, Gillian
CS School of Biological Sciences, University of East Anglia, Norwich, NR4
7TJ, UK
SO Journal of Biological Chemistry (1999), 274(29), 20391-20396
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 46 OF 62 MEDLINE on STN DUPLICATE 29
AN 1999282197 MEDLINE
DN PubMed ID: 10353844
TI The N-terminus of collagenase **MMP**-8 determines superactivity and **inhibition**: a relation of structure and function analyzed by biomolecular interaction analysis.
AU Farr M; Pieper M; Calvete J; Tschesche H
CS University of Bielefeld, Faculty of Chemistry and Biochemistry, Germany.
SO Biochemistry, (1999 Jun 1) 38 (22) 7332-8.
Journal code: 0370623. ISSN: 0006-2960.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199906
ED Entered STN: 19990712
Last Updated on STN: 20000303
Entered Medline: 19990623

L5 ANSWER 47 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2000:152140 CAPLUS
DN 133:68500
TI Dibutyryl cyclic AMP-induced enhancement of tissue inhibitor of metalloproteinases-3 expression and its possible relation to the invasive
activity of the human hepatoma cell line PLC/PRF/5
AU Okamoto, Yasuyuki; Nakano, Hiroshi
CS Department of Clinico-Laboratory Diagnostics, Nara Medical University,
Nara, 634-8521, Japan
SO Anticancer Research (1999), 19(6B), 5175-5180
CODEN: ANTRD4; ISSN: 0250-7005
PB International Institute of Anticancer Research
DT Journal
LA English
RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 48 OF 62 MEDLINE on STN DUPLICATE 30

AN 1999257345 MEDLINE
DN PubMed ID: 10323763
TI Role of matrix metalloproteinases and their tissue inhibitors in the
regulation of coronary cell migration.
AU Shi Y; Patel S; Niculescu R; Chung W; Desrochers P; Zalewski A
CS Cardiovascular Research Center, Department of Medicine (Cardiology),
Thomas Jefferson University, Philadelphia, PA 19107, USA..
yi.shi@mail.tju.edu
NC HL-44150 (NHLBI)
HL-60672 (NHLBI)
SO Arteriosclerosis, thrombosis, and vascular biology, (1999 May) 19 (5)
1150-5.
Journal code: 9505803. ISSN: 1079-5642.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199906
ED Entered STN: 19990618
Last Updated on STN: 20000303
Entered Medline: 19990607

L5 ANSWER 49 OF 62 MEDLINE on STN DUPLICATE 31
AN 1999344222 MEDLINE
DN PubMed ID: 10415733
TI Measurement of matrix metalloproteinases and tissue inhibitors of
metalloproteinases in blood and tissues. Clinical and experimental
applications.
AU Zucker S; Hymowitz M; Conner C; Zarrabi H M; Hurewitz A N; Matrisian L;
Boyd D; Nicolson G; Montana S
CS Department of Medicine and Research, Veterans Administration Medical
Center, Northport, New York 11768, USA..
zucker.stanley@northport.va.gov
SO Annals of the New York Academy of Sciences, (1999 Jun 30) 878 212-27.
Ref: 75
Journal code: 7506858. ISSN: 0077-8923.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199908
ED Entered STN: 19990820
Last Updated on STN: 20000303

Entered Medline: 19990811

L5 ANSWER 50 OF 62 MEDLINE on STN DUPLICATE 32
AN 1999114750 MEDLINE
DN PubMed ID: 9918218
TI Interleukin 10 (IL-10) inhibition of primary human prostate cell-induced
angiogenesis: IL-10 stimulation of tissue inhibitor of metalloproteinase-1
and **inhibition** of matrix metalloproteinase (**MMP**)-2/MMP-9 secretion.
AU Stearns M E; Rhim J; Wang M
CS Department of Pathology and Laboratory Sciences, Medical College of
Pennsylvania and Hahnemann University, Philadelphia 19102-1192, USA.
SO Clinical cancer research : an official journal of the American Association
for Cancer Research, (1999 Jan) 5 (1) 189-96.
Journal code: 9502500. ISSN: 1078-0432.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199903
ED Entered STN: 19990402
Last Updated on STN: 20000303
Entered Medline: 19990324

L5 ANSWER 51 OF 62 MEDLINE on STN DUPLICATE 33
AN 1999023989 MEDLINE
DN PubMed ID: 9804828
TI Differential expression of aggrecanase and matrix metalloproteinase
activity in chondrocytes isolated from bovine and porcine articular
cartilage.
AU Hughes C E; Little C B; Buttner F H; Bartnik E; Caterson B
CS Connective Tissue Biology Laboratories, Cardiff School of Biosciences,
Cardiff University, Cardiff CF1 3US, Wales, United Kingdom..
hughesce1@cardiff.ac.uk
SO Journal of biological chemistry, (1998 Nov 13) 273 (46) 30576-82.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199812
ED Entered STN: 19990115
Last Updated on STN: 20000303

Entered Medline: 19981208

L5 ANSWER 52 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1998:254786 CAPLUS
DN 129:53243
TI IL-10 inhibition of human prostate PC-3 ML cell metastases in SCID mice:
IL-10 stimulation of **TIMP**-1 and **inhibition** of **MMP**-2/**MMP**-9 expression
AU Stearns, M. E.; Fudge, K.; Garcia, F.; Wang, M.
CS Department of Pathology and Laboratory Medicine, Allegheny University,
Philadelphia, PA, 19102-1192, USA
SO Invasion & Metastasis (1998), Volume Date 1997, 17(2), 62-74
CODEN: INVMDJ; ISSN: 0251-1789
PB S. Karger AG
DT Journal
LA English
RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 53 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1997:345955 CAPLUS
DN 127:60267
TI Inhibition of osteolytic bone metastasis of breast cancer by combined
treatment with the bisphosphonate ibandronate and tissue inhibitor of the
matrix metalloproteinase-2
AU Yoneda, Toshiyuki; Sasaki, Akira; Dunstan, Colin; Williams, Paul J.;
Bauss, Frieder; De Clerck, Yves A.; Mundy, Gregory R.
CS Department of Medicine, Division of Endocrinology and Metabolism,
University of Texas Health Science Center at San Antonio, San Antonio, TX,
78284-7877, USA
SO Journal of Clinical Investigation (1997), 99(10), 2509-2517
CODEN: JCINAO; ISSN: 0021-9738
PB Rockefeller University Press
DT Journal
LA English
RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 54 OF 62 MEDLINE on STN DUPLICATE 34
AN 1998008087 MEDLINE
DN PubMed ID: 9344510
TI TIMP-3 induces cell death by stabilizing TNF-alpha receptors on the
surface of human colon carcinoma cells.
AU Smith M R; Kung H; Durum S K; Colburn N H; Sun Y
CS Intramural Research Support Program, SAIC Frederick, National Cancer

Institute, Frederick Cancer Research and Development Center, Frederick, MD
21702, USA.
SO Cytokine, (1997 Oct) 9 (10) 770-80.
Journal code: 9005353. ISSN: 1043-4666.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199801
ED Entered STN: 19980130
Last Updated on STN: 19980130
Entered Medline: 19980122

L5 ANSWER 55 OF 62 MEDLINE on STN DUPLICATE 35
AN 1998221796 MEDLINE
DN PubMed ID: 9561025
TI IL-10 inhibition of human prostate PC-3 ML cell metastases in SCID mice:
IL-10 stimulation of **TIMP**-1 and **inhibition** of **MMP**-2/**MMP**-9 expression.
AU Stearns M E; Fudge K; Garcia F; Wang M
CS Department of Pathology and Laboratory Medicine, Allegheny University,
Philadelphia, Pa. 19102-1192, USA.
SO Invasion & metastasis, (1997) 17 (2) 62-74.
Journal code: 8202435. ISSN: 0251-1789.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199805
ED Entered STN: 19980514
Last Updated on STN: 20000303
Entered Medline: 19980505

L5 ANSWER 56 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1998:177079 CAPLUS
DN 128:293277
TI Tissue inhibitors of metalloproteinases and metastasis
AU Iwata, Hiroji; Kobayashi, Syunzo
CS Second Dep. Surg., Nagoya City Univ. Sch. Med., Japan
SO Ketsueki, Men'eki, Shuyo (1996), 1(4), 348-355
CODEN: KMSHF6; ISSN: 1341-5824
PB Medikaru Rebyusha
DT Journal; General Review
LA Japanese

L5 ANSWER 57 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1996:648357 CAPLUS
DN 126:58732
TI Interleukin-4 blocks the release of collagen fragments from bovine nasal

cartilage treated with cytokines
AU Cawston, Tim E.; Ellis, Alison J.; Bigg, Heather; Curry, Valerie; Lean,
Eileen; Ward, Dawn
CS Rheumatology Research Unit, Box 194, Addenbrooke's Hospital, Cambridge,
CB2 2QQ, UK
SO Biochimica et Biophysica Acta, Molecular Cell Research (1996), 1314(3),
226-232
CODEN: BBAMCO; ISSN: 0167-4889
PB Elsevier B.V.
DT Journal
LA English

L5 ANSWER 58 OF 62 MEDLINE on STN DUPLICATE 36
AN 95355479 MEDLINE
DN PubMed ID: 7629179
TI Steps involved in activation of the pro-matrix metalloproteinase 9
(progelatinase B)-tissue inhibitor of metalloproteinases-1 complex by
4-aminophenylmercuric acetate and proteinases.
AU Ogata Y; Itoh Y; Nagase H
CS Department of Biochemistry and Molecular Biology, University of Kansas
Medical Center, Kansas City 66160-7421, USA.
NC AR39189 (NIAMS)
AR40994 (NIAMS)
SO Journal of biological chemistry, (1995 Aug 4) 270 (31) 18506-11.

Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199509
ED Entered STN: 19950921
Last Updated on STN: 20000303
Entered Medline: 19950907

L5 ANSWER 59 OF 62 MEDLINE on STN DUPLICATE 37
AN 95301511 MEDLINE
DN PubMed ID: 7782289
TI The gene structure of tissue inhibitor of metalloproteinases (TIMP)-3 and
its inhibitory activities define the distinct TIMP gene family.
CM Erratum in: J Biol Chem. 1996 Feb 2;271(5):2874. PubMed ID: 8576269
AU Apte S S; Olsen B R; Murphy G
CS Department of Cell Biology, Harvard Medical School, Boston, Massachusetts

02115, USA.
NC AR36819 (NIAMS)
AR36820 (NIAMS)
HL33014 (NHLBI)
+
SO Journal of biological chemistry, (1995 Jun 16) 270 (24) 14313-8.

Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U26433; GENBANK-U26434; GENBANK-U26435; GENBANK-U26436; GENBANK-U26437
EM 199507
ED Entered STN: 19950726
Last Updated on STN: 20000303
Entered Medline: 19950717

L5 ANSWER 60 OF 62 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on
STN
AN 1995:97255 BIOSIS
DN PREV199598111555
TI Role of **TIMP** and **MMP inhibition** in
preventing connective tissue breakdown.
AU Cawston, Tim; Plumpton, Tracy; Curry, Valerie; Ellis, Alison; Powell, Liz
CS Rheumatology Res. Unit, Box 194, Addenbrookes Hosp., Hill's Road, Cambridge CB2 2QQ, UK
SO Greenwald, R. A. [Editor]; Golub, L. M. [Editor]. Ann. N. Y. Acad. Sci.,
(1994) pp. 75-83. Annals of the New York Academy of Sciences; Inhibition
of matrix metalloproteinases: Therapeutic potential.
Publisher: New York Academy of Sciences, 2 East 63rd Street, New York, New
York 10021, USA. Series: Annals of the New York Academy of Sciences.
Meeting Info.: Conference. Tampa, Florida, USA. January 19-22, 1994.
CODEN: ANYAA9. ISSN: 0077-8923. ISBN: 0-89766-900-2 (paper), 0-89766-899-5
(cloth).
DT Book
Conference; (Meeting)
Book; (Book Chapter)
Conference; (Meeting Paper)
LA English
ED Entered STN: 1 Mar 1995
Last Updated on STN: 1 Mar 1995

L5 ANSWER 61 OF 62 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1995:313189 CAPLUS
DN 123:4253
TI Characterization of tissue inhibitor of metalloproteinases 2 (TIMP-2) from
human hepatoma cell line (HLE cells) and its interaction with gelatinase A
and inactivation
AU Kawamura, Kouji
CS School Medicine, Kanazawa Univ., Kanazawa, 920, Japan
SO Kanazawa Daigaku Juzen Igakkai Zasshi (1994), 103(4), 690-700
CODEN: JUZIAG; ISSN: 0022-7226
PB Juzen Igakkai
DT Journal
LA Japanese

L5 ANSWER 62 OF 62 MEDLINE on STN DUPLICATE 38
AN 95069560 MEDLINE
DN PubMed ID: 7978853
TI Role of **TIMP** and **MMP inhibition** in
preventing connective tissue breakdown.
AU Cawston T; Plumpton T; Curry V; Ellis A; Powell L
CS Rheumatology Research Unit, Addenbrookes Hospital, Cambridge, United
Kingdom.
SO Annals of the New York Academy of Sciences, (1994 Sep 6) 732 75-83.
Journal code: 7506858. ISSN: 0077-8923.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199412
ED Entered STN: 19950110
Last Updated on STN: 20000303
Entered Medline: 19941202

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of CAplus documents for use in third-party analysis and
visualization tools
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(4A)(inhibition or inhibit or inhibiting or inhibited)
L1          1846 (TIMP OR (TISSUE INHIBITORS OF METALLOPROTEINASES))
(4A)(INHIBIT
                 ION OR INHIBIT OR INHIBITING OR INHIBITED)

=> s ((Matrix metalloproteinase)  or MMP)(4A)(inhibition or inhibit
or inhibiting or inhibited)
L2          8293 ((MATRIX METALLOPROTEINASE) OR MMP)(4A)(INHIBITION OR
INHIBIT
                 OR INHIBITING OR INHIBITED)

=> S L1 AND L2
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L3          669 L1 AND L2

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L4           23 L3 AND (2001 OR 2000 OR 1999 OR 1998)

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L5   ANSWER 1 OF 15     MEDLINE on STN                    DUPLICATE 1
AN   2004509808     MEDLINE
DN   PubMed ID: 15467768
TI   An immunohistochemical study of TIMP-3 expression in oesophageal
squamous
     cell carcinoma.
AU   Miyazaki T; Kato H; Nakajima M; Faried A; Takita J; Sohda M;
Fukai Y;
     Yamaguchi S; Masuda N; Manda R; Fukuchi M; Ojima H; Tsukada K;
Kuwano H
CS   Department of General Surgical Science (Surgery I), Gunma
University
     Graduate School of Medicine, 3-39-22, Showa-machi, Maebashi,
Gunma
     371-8511, Japan.. tatsuyam@showa.gunma-u.ac.jp
SO   British journal of cancer, (2004 Oct 18) 91 (8) 1556-60.
     Journal code: 0370635. ISSN: 0007-0920.
CY   England: United Kingdom
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   200411
ED   Entered STN: 20041014
     Last Updated on STN: 20041117
     Entered Medline: 20041116
AB   Tissue inhibitor of metalloproteinase-3 (TIMP-3)
     inhibits the activity of matrix
     metalloproteinase, which may play an important role in carcinoma
     invasion and metastasis.  We have investigated the relationship
between
     TIMP-3 reduction and clinicopathological factors in oesophageal
squamous
     cell carcinoma (ESCC).  We examined tissue specimens that had
been removed
     from 90 patients with thoracic oesophageal cancer who had
undergone
     surgery between 1983 and 2001.  Immunohistochemical staining was
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performed by the standard streptavidin-biotin method. Immunostaining of TIMP-3 was seen in the cytoplasm of cancer cells and normal oesophageal epithelial cells, particularly in cells located in shallow areas of the tumour. TIMP-3 preserved (+), moderate (+/-), and reduced (-) cases accounted for 30, 27, and 33 of the 90 patients, respectively (33, 30, 37%). Significant correlations were observed between TIMP-3 expression and depth of tumour invasion (P=0.001), number of lymph node metastases (P=0.003), infiltrative growth pattern (P=0.003), and disease stage (P=0.005). The survival rates of patients with TIMP-3 (-) cancer were significantly lower than those of patients with TIMP-3 (+) and TIMP-3 (+/-) cancer (P=0.0003). The mean 5-year survival rates of patients with TIMP-3 (+), (+/-), and (-) were 50, 58, and 21%, respectively. In conclusion, decreased expression of TIMP-3 protein correlates with invasive activity and metastasis. This makes the prognosis for patients with cancer that has lost TIMP-3 significantly less favourable than that for patients with cancer that has maintained TIMP-3.

AN 2002314329 EMBASE

TI Dexamethasone inhibits vascular smooth muscle cell migration via modulation of matrix metalloproteinase activity.

AU Pross C.; Farooq M.M.; Angle N.; Lane J.S.; Cerveira J.J.; Xavier A.E.; Freischlag J.A.; Law R.E.; Gelabert H.A.

CS Dr. H.A. Gelabert, Medical Plaza 200, Los Angeles, CA 90095, United States. hgelabert@mednet.ucla.edu

SO Journal of Surgical Research, (2002) Vol. 102, No. 2, pp. 57-62. Refs: 28
ISSN: 0022-4804 CODEN: JSGRA2

CY United States

DT Journal; Article

FS 009 Surgery
018 Cardiovascular Diseases and Cardiovascular Surgery
030 Pharmacology

037 Drug Literature Index
LA English
SL English
ED Entered STN: 20020919
Last Updated on STN: 20020919
AB Background. Dexamethasone (DEX) has been shown to inhibit development of neointimal hyperplasia in rats. We hypothesize that DEX **inhibits** neointimal hyperplasia by altering **matrix metalloproteinase** (**MMP**) activity, resulting in **inhibition** of smooth muscle cell migration. Methods. Rat aortic smooth muscle cells (RASMC) were harvested and cultured for two to four passages. A migration assay was performed in a Boyden chamber with chemoattractant (platelet-derived growth factor) and varying concentrations of DEX (10(-9) to 10(-5) M). The number of migrated cells was counted under light microscopy. Zymography was performed on culture media to assess MMP activity, and Western blotting was performed to assay MMP and levels of tissue inhibitors of MMPs (TIMPs). Results. DEX progressively inhibited RASMC migration in a dose-dependent fashion. This effect was statistically significant for concentrations of 10(-7) to 10(-5) M ($P < 0.0005$). Zymography showed that DEX **inhibits** **MMP**-2 activity in a dose-dependent manner. Western blots indicated that total **MMP**-2 secretion was **inhibited** and that **TIMP**-2 secretion was increased by DEX. Conclusions. DEX inhibits platelet-derived growth factor-induced migration of RASMCs and MMP-2 activity in vitro. Our data suggest that DEX suppresses MMP activity and secretion, resulting in the inhibition of smooth muscle cell migration. This may explain the mechanism by which DEX inhibits neointimal hyperplasia. .COPYRGT. **2001** Elsevier Science.

L5 ANSWER 3 OF 15 EMBASE
AN 2001076223 EMBASE
TI Effects of nitric oxide on matrix metalloproteinase-2 production by rheumatoid synovial cells.
AU Hirai Y.; Migita K.; Honda S.; Ueki Y.; Yamasaki S.; Urayama S.; Kamachi M.; Kawakami A.; Ida H.; Kita M.; Fukuda T.; Shibatomi K.; Kawabe Y.;

Aoyagi T.; Eguchi K.
CS K. Eguchi, First Dept. of Internal Medicine, Nagasaki Univ. School of Medicine, Sakamoto 1-7-1, Nagasaki 852-8501, Japan
SO Life Sciences, (12 Jan 2001) Vol. 68, No. 8, pp. 913-920.
Refs: 22
ISSN: 0024-3205 CODEN: LIFSAK
PUI S 0024-3205(00)00998-X
CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
031 Arthritis and Rheumatism
LA English
SL English
ED Entered STN: 20010316
Last Updated on STN: 20010316
AB Nitric oxide (NO) is a multifunctional messenger molecule generated from L-arginine by a family of enzymes, including nitric oxide synthase (NOS).
This study was performed to examine whether NO modulates the production of matrix metalloproteinases (MMPs), which degrade all components of extracellular matrix (ECM), in rheumatoid synovial cells. We investigated the effects of exogenously generated NO by a NO donor, S-nitroso-N-acetyl-DL-penicillamine (SNAP), on the MMPs production by rheumatoid synovial cells. Culture media conditioned by SNAP-treated synovial cells were examined by gelatin zymography and immunoblot analysis. Incubation of synovial cells with SNAP resulted in gelatinase A production in a dose-dependent fashion. Furthermore, RT-PCR analysis demonstrated that MMP-2 mRNA expression was induced in SNAP-treated synovial cells. In contrast, SNAP did not influence the production of TIMP-1 and **TIMP**-2, which preferentially **inhibit** **MMP**-2, by rheumatoid synovial cells. Our data indicate that NO could modulate MMP production by rheumatoid synovial cells and therefore contribute to ECM degradation of articular components in RA. .COPYRGT.
2001 Elsevier Science Inc.

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AN 2001240567 EMBASE
TI High glucose decreases matrix metalloproteinase-2 activity in rat

mesangial cells via transforming growth factor-β(1).
AU Singh R.; Ruo Hua Song; Alavi N.; Pegoraro A.A.; Singh A.K.; Leehey D.J.
CS Dr. D.J. Leehey, Veterans Affairs Hospital, Hines, IL 60141, United States. david.leehey@med.va.gov
SO Experimental Nephrology, (2001) Vol. 9, No. 4, pp. 249-257.
Refs: 32
ISSN: 1018-7782 CODEN: EXNEEG
CY Switzerland
DT Journal; Article
FS 003 Endocrinology
028 Urology and Nephrology
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 20010726
Last Updated on STN: 20010726
AB Diabetic nephropathy is characterized by accumulation of mesangial matrix.
Glucose-induced inhibition of matrix-degrading enzymes such as collagenases is believed to contribute to matrix accumulation. We have previously demonstrated that 72 kDa type IV collagenase activity is decreased in the rat mesangial cells cultured in high glucose media [Diabetes 1995;44:929-935]. The present studies were designed to investigate if the cytokine transforming growth factor-β(1) (TGF-β(1)) mediates this effect of glucose. Type IV collagenases degrade type IV collagen as well as gelatin (denatured collagen) and are thus also called gelatinases. They belong to the family of matrix metalloproteinases (MMPs); MMP activity is controlled by tissue inhibitors of metalloproteinases (TIMPs). The activity of 72 kDa type IV collagenase, also known as matrix metalloproteinase-2 (MMP-2), was assessed using three methods: (1) fluoresceinated gelatin degradation assay to detect free enzyme activity (activity which is present in excess of **TIMP-inhibited** activity); (2) zymography to measure total (free + TIMP-bound) enzyme activity; (3) ELISA using specific antibodies to measure MMP-2 levels. TGF-β(1) and TIMP-2 levels were)also determined by ELISA. Incubation of primary cultures of rat mesangial cells for 5 days in 30 vs. 5 mM glucose resulted in a 3-fold

increase in production of total TGF-β(1), a significant decrease in MMP-2 activity and immunoreactive MMP-2 levels, and an increase in TIMP-2 levels. Addition of exogenous TGF-β(1) to mesangial cells incubated in 5 mM glucose replicated the high glucose effect by producing a significant decrease in MMP-2 levels with a concurrent increase in TIMP-2 levels. Furthermore, glucose-induced **inhibition** of **MMP** -2 activity was completely blocked by neutralization of TGF-β(1) with anti-TGF-β(1) antibody. We conclude that the decrease in MMP-2 activity induced by glucose loading is mediated via TGF-β(1).

L5 ANSWER 5 OF 15 EMBASE

AN 2001016268 EMBASE

TI **Inhibition** of **matrix metalloproteinases** by over-expression of tissue inhibitor of metalloproteinase-2 inhibits the growth of experimental hemangiomas.

AU Vergani V.; Garofalo A.; Bani M.R.; Borsotti P.; Parker M.P.; Drudis T.; Mazzarol G.; Viale G.; Giavazzi R.; Stetler-Stevenson W.G.; Taraboletti G.

CS G. Taraboletti, Department of Oncology, Mario Negri Inst. of Pharmacol. Res., via Gavazzeni 11, 24125 Bergamo, Italy. taraboletti@irfmn.mnegri.it

SO International Journal of Cancer, (15 Jan 2001) Vol. 91, No. 2, pp. 241-247.
Refs: 39
ISSN: 0020-7136 CODEN: IJCNAW

CY United States

DT Journal; Article

FS 016 Cancer
030 Pharmacology
037 Drug Literature Index

LA English

SL English

ED Entered STN: 20010125
Last Updated on STN: 20010125

AB Inhibitors of proteases prevent tumor-associated matrix degradation, affecting tumor growth, angiogenesis and metastasis. Our study was designed to investigate the effect of **inhibition** of **matrix metalloproteinases** (**MMPs**) on the growth

of experimental hemangiomas, using the model of murine endothelioma eEnd.1 cells. In nude mice, these cells generate hemangiomas, consisting mostly of host-recruited endothelial cells, whose growth requires the activity of MMPs. In vitro, eEnd.1 cells produce factors that recruit endothelial cells and stimulate them to release MMPs. Over-expression of TIMP-2, following retrovirus-mediated gene transfer, decreased tumor growth in vivo. The infected clone CR1, which produces high levels of TIMP-2 (as assessed by Northern blot, ELISA and reverse zymography), formed slow-growing tumors that did not grow beyond 0.4 g, while clone IH, which produces little TIMP-2, grew not dissimilarly to mock-infected cells and parental e.End.1 cells. Histologically, control tumors presented the features of cavernous hemangiomas, while CR1 tumors had a more solid pattern, showing fool of apoptotic cells. In vitro, TIMP-2 over-expression had no autocrine anti-proliferative effect on endothelioma cells but reduced their ability to recruit endothelial cells. CR1 cells lacked the capacity of mock-infected or parental eEnd.1 cells to stimulate endothelial cell motility and invasiveness. Antibodies against TIMP-2 restored the ability of CR1 to induce endothelial cell invasion. We conclude that, in this model, genetic increase of **TIMP**-2 **inhibits** tumor growth, apparently by affecting the recruitment and organization of host endothelial cells by the transformed cells. .COPYRGT. **2001** Wiley-Liss, Inc.

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AN 2001246293 EMBASE

TI CD40L induces matrix-metalloproteinase-9 but not tissue inhibitor of metalloproteinases-1 in cervical carcinoma cells: Imbalance between NF-κB and STAT3 activation.

AU Smola-Hess S.; Schnitzler R.; Hadaschik D.; Smola H.; Mauch C.; Krieg T.;

Pfister H.

CS S. Smola-Hess, Institute of Virology, University of Cologne, Furst-Puckler-Str. 56, 50935 Cologne, Germany. s.hess@ni-koeln.de

SO Experimental Cell Research, (15 Jul 2001) Vol. 267, No. 2, pp. 205-215.
Refs: 76
ISSN: 0014-4827 CODEN: ECREAL
CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 20010726
Last Updated on STN: 20010726

AB Matrix-metalloproteinases (MMPs) are essentially required for tumor cell invasion and metastasis. Production of precursor enzymes is regulated on transcriptional level, while activation of the pro-enzymes is tightly controlled by posttranscriptional mechanisms. The enzyme activity can be blocked by specific tissue inhibitors of MMPs (TIMPs). In cervical carcinomas strong up-regulation of the type IV collagenase MMP-9 had been demonstrated. We show that activation of CD40, a receptor highly expressed on cervical carcinomas, induces MMP-9 in cervical carcinoma cells, whereas **TIMP**-1 production **inhibiting** **MMP**-9 activity was not affected. This gene induction pattern corresponded to the differential activation of the transcription factor nuclear factor κB (NF-κB) regulating MMP-9, but not signal transducer and activator of transcription 3 (STAT3), which is involved in TIMP-1 gene regulation. Transient expression of the CD40-inducible NF-κB subunit p65 was sufficient for MMP-9 induction. Agents that suppressed CD40-mediated NF-κB activation also reduced MMP-9 induction, further supporting an important role of NF-κB in CD40-mediated MMP-9 induction. Our data suggest that CD40 expression in carcinoma cells might convert a CD40L-dependent immunological defense signal into a tumor-promoting signal. Selective CD40-mediated signaling through NF-κB but not STAT3 correlates to a shift of the balance between MMP-9 and TIMP-1 toward the protease. .COPYRGT. **2001** Academic Press.

L5 ANSWER 7 OF 15 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights
reserved on STN DUPLICATE 3
AN 2001195366 EMBASE
TI Type I collagen stabilization of matrix metalloproteinase-2.
AU Ellerbroek S.M.; Wu Y.I.; Stack M.S.
CS M.S. Stack, Northwestern Univ. Medical School, Department of Cell, 303 E.
Chicago Avenue, Chicago, IL 60611, United States.
mss130@northwestern.edu
SO Archives of Biochemistry and Biophysics, (1 Jun 2001) Vol. 390, No. 1, pp.
51-56.
Refs: 35
ISSN: 0003-9861 CODEN: ABBIA4
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 20010614
Last Updated on STN: 20010614
AB The activity of matrix metalloproteinase-2 (MMP-2) is regulated stringently on the posttranslational level. MMP-2 efficiently undergoes autolysis into inactive polypeptides in vitro, prompting the hypothesis that MMP-2 autolysis may function as an alternative mechanism for posttranslational control of MMP-2 in vivo. Moreover, MMP-2 binds to intact type I collagen fibrils; however, the functional consequences of this interaction have not been fully elucidated. To test the hypothesis that MMP-2 binding to type I collagen functions as a positive regulator of MMP-2 proteolytic potential, the effect of type I collagen on **MMP**-2 activity, **inhibition** by tissue inhibitor of metalloproteinase-2 (TIMP-2), and enzyme stability was examined. Here, we report that purified MMP-2 binds but does not cleave intact type I collagen. The presence of type I collagen affects neither enzymatic activity against a quenched fluorescent peptide substrate nor the kinetics of **inhibition** by **TIMP**-2. However, MMP-2 is stabilized from autolysis in the presence of type I collagen, but not by elastin, fibrinogen, or laminin. These data provide biochemical evidence that

MMP.2 exosite interactions with type I collagen may function in the posttranslational control of MMP-2 activity by reducing the rate of autolytic inactivation. .COPYRGT. **2001** Academic Press.

L5 ANSWER 8 OF 15 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN DUPLICATE 4

AN 2000228169 EMBASE

TI Regulation and significance of hepatocyte-derived matrix metalloproteinases in liver remodeling.

AU Haruyama T.; Ajioka I.; Akaike T.; Watanabe Y.

CS Y. Watanabe, Department Biomolecular Engineering, Tokyo Institute of Technology, 4259 Nagatsuda, Midori-ku, Yokohama 226-850, Japan. ywatanab@bio.titech.ac.jp

SO Biochemical and Biophysical Research Communications, (16 Jun 2000) Vol. 272, No. 3, pp. 681-686.
Refs: 36
ISSN: 0006-291X CODEN: BBRCA

CY United States

DT Journal; Article

FS 029 Clinical Biochemistry
048 Gastroenterology

LA English

SL English

ED Entered STN: 20000720
Last Updated on STN: 20000720

AB Regulation in expression and activation of proteinases is one of the most important mechanisms in organ morphogenesis. In this study, we investigated the expression of MMPs in primary hepatocytes and their roles in liver remodeling. A hepatocyte proliferation initiating cytokine, TNFα, induced MMP-9 expression in these cells while the expression of MMP-2 did not change by zymography analysis. Interestingly, both the induced MMP-9 expression and hepatocyte proliferation by TNFα were synergistically enhanced by HGF in vitro. The increased proliferation was suppressed by MMP inhibitor TIMP-1, suggesting that cytokine-induced MMP regulates proliferation. The increased expression of MMP-9 by the cytokines was inhibited by cytochalasin D or colchicine but not by PI3 kinase inhibitor wortmannin. In addition, costimulation by TNFα and

HGF of spheroidal hepatocytes cultured in 3-dimensional collagen gel drastically induced morphological changes by cell extension and migration in the gel, which was in parallel with the induced expression of **MMP**-9 and was **inhibited** by **TIMP**-1 and -2. The MMP activity was also detected in vivo in the regenerating liver after partial hepatectomy by in situ zymography. These results suggest the roles of MMPs produced by parenchymal cells in liver remodeling. (C) **2000** Academic Press.

L5 ANSWER 9 OF 15 EMBASE
AN 2000383693 EMBASE
TI Prognostic impact of tissue inhibitor of matrix metalloproteinase-1 in esophageal carcinoma.
AU Mori M.; Mimori K.; Sadanaga N.; Inoue H.; Tanaka Y.; Mafune K.-I.; Ueo H.; Barnard G.F.
CS M. Mori, Department of Surgery, Medical Institute of Bioregulation, Kyushu University, 4546 Tsurumibaru, Beppu 874-0838, Japan. mmori@beppu.kyushu-u.ac.jp
SO International Journal of Cancer, (2000) Vol. 88, No. 4, pp. 575-578.
Refs: 27
ISSN: 0020-7136 CODEN: IJCNAW
CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
011 Otorhinolaryngology
016 Cancer
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 20001213
Last Updated on STN: 20001213
AB Tissue inhibitor of metalloproteinase-1 (**TIMP**-1) **inhibits** the activity of **matrix metalloproteinase**, which may play an important role in carcinoma invasion and metastasis. TIMP-1 is thus considered to inhibit carcinoma invasion and metastasis. However, TIMP-1 possesses another important function, cell growth promotion. The clinical significance of TIMP-1

expression has not been fully determined in esophageal carcinoma. We thus examined the expression of TIMP-1 mRNA in tumor (T) and corresponding normal (N) tissues of 85 esophageal carcinoma cases by RT-PCR. The T:N ratio of TIMP-1 mRNA expression in each case was evaluated semiquantitatively with adjustment by an internal control gene. The mean T:N ratio was 2.0 (range 0.2-6.5). When comparing high-expression cases (T:N > 2.0, n = 37) with low-expression cases (T:N ≤ 2.0, n = 48), the former showed a significantly higher frequency of lymph vessel invasion, vascular vessel invasion, lymph node metastasis and advanced-stage disease. The former cases showed a poorer prognosis than the latter. Multivariate analysis disclosed that TIMP-1 expression status was an independent determining factor for prognosis. Our findings suggest that TIMP-1 expression correlates with tumor extension of esophageal carcinoma and might, if validated, prove useful as a novel prognostic marker for esophageal carcinoma. (C) **2000** Wiley-Liss, Inc.

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AN 2000281993 EMBASE

TI Regulation of tissue inhibitor of metalloproteinase-3 (Timp-3) mRNA expression during rat CNS development.

AU Jaworski D.M.; Fager N.

CS Dr. D.M. Jaworski, Dept. of Anatomy/Neurobiology, Univ. of Vermont College of Medicine, Given C454, Burlington, VT 05405, United States. djaworsk@zoo.uvm.edu

SO Journal of Neuroscience Research, (15 Aug 2000) Vol. 61, No. 4, pp. 396-408.
Refs: 77
ISSN: 0360-4012 CODEN: JNREDK

CY United States

DT Journal; Article

FS 008 Neurology and Neurosurgery
029 Clinical Biochemistry

LA English

SL English

ED Entered STN: 20000831

Last Updated on STN: 20000831

AB To preserve tissue integrity during the structural rearrangements that occur during central nervous system (CNS) development, an intricate balance between extra-cellular matrix (ECM) synthesis and degradation must be maintained. The matrix metalloproteinases (MMPs) are believed to be the main mediators of ECM degradation. Because MMPs function in the turnover of a broad-spectrum of ECM proteins their activity is tightly regulated by interaction with tissue inhibitors of metalloproteinases (TIMPs). Whereas the primary function of **TIMPs** is to **inhibit MMP** activity, evidence is mounting that TIMPs are multifunctional molecules that exert diverse cell biological functions distinct from their MMP-inhibitory activities. Although the role of MMPs and TIMPs in the morphogenesis of non-neural tissues has been investigated, to date few studies have analyzed MMP or TIMP expression during CNS development. In the present report, we demonstrate the regulation of Timp-3 mRNA expression throughout the course of CNS development. In particular, Timp-3 mRNA is expressed in embryonic ventricular zones and the postnatal subventricular zone (SVZ). In addition, Timp-3 is expressed in the rostral migratory steam (RMS) to the olfactory bulb in a pattern similar to the ECM proteoglycan brevican. These data suggest that TIMP-3 and brevican may act in concert to guide neuronal migration along the RMS. (C) **2000** Wiley-Liss, Inc.

L5 ANSWER 11 OF 15 EMBASE

AN 2000081145 EMBASE

TI Tissue inhibitors of metalloproteinases: Evolution, structure and function.

AU Brew K.; Dinakarpandian D.; Nagase H.

CS K. Brew, Department of Biochemistry, University of Miami, School of Medicine, Miami, FL 33101, United States. kbrew@molbio.med.miami.edu

SO Biochimica et Biophysica Acta - Protein Structure and Molecular Enzymology, (7 Mar 2000) Vol. 1477, No. 1-2, pp. 267-283.

Refs: 74
ISSN: 0167-4838 CODEN: BBAEDZ
PUI S 0167-4838(99)00279-4
CY Netherlands
DT Journal; General Review
FS 029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 20000316
Last Updated on STN: 20000316
AB The matrix metalloproteinases (MMPs) play a key role in the normal physiology of connective tissue during development, morphogenesis and wound healing, but their unregulated activity has been implicated in numerous disease processes including arthritis, tumor cell metastasis and atherosclerosis. An important mechanism for the regulation of the activity of MMPs is via binding to a family of homologous proteins referred to as the tissue inhibitors of metalloproteinases (TIMP-1 to TIMP-4). The two-domain TIMPs are of relatively small size, yet have been found to exhibit several biochemical and physiological/biological functions, including **inhibition** of active **MMPs**, proMMP activation, cell growth promotion, matrix binding, inhibition of angiogenesis and the induction of apoptosis. Mutations in TIMP-3 are the cause of Sorsby's fundus dystrophy in humans, a disease that results in early onset macular degeneration. This review highlights the evolution of TIMPs, the recently elucidated high-resolution structures of TIMPs and their complexes with metalloproteinases, and the results of mutational and other studies of structure-function relationships that have enhanced our understanding of the mechanism and specificity of the **inhibition** of **MMPs** by **TIMPs**. Several intriguing questions, such as the basis of the multiple biological functions of TIMPs, the kinetics of TIMP-MMP interactions and the differences in binding in some TIMP-metalloproteinase pairs are discussed which, though not fully resolved, serve to illustrate the kind of issues that are important for a full understanding of the interactions between families of molecules.

L5 ANSWER 12 OF 15 EMBASE
AN 2000434576 EMBASE
TI TIMP-1/MMP-9 imbalance in an EBV-immortalized B lymphocyte cellular model:
Evidence for TIMP-1 multifunctional properties.
AU Gaudin P.; Trocme C.; Berthier S.; Kieffer S.; Boutonnat J.; Lamy C.;
Surla A.; Garin J.; Morel F.
CS F. Morel, GREPI, Laboratoire d'Enzymologie, CHU A. Michallon, BP 217,
38043 Grenoble Cedex 9, France. frmorel.enzymo@chu-grenoble.fr
SO Biochimica et Biophysica Acta - Molecular Cell Research, (11 Dec 2000)
Vol. 1499, No. 1-2, pp. 19-33.
Refs: 46
ISSN: 0167-4889 CODEN: BAMRDP
PUI S 0167-4889(00)00084-7
CY Netherlands
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 20010119
Last Updated on STN: 20010119
AB Tissue inhibitors of metalloproteinases (TIMPs) were initially described
as agents controlling metalloproteinase activity. The purpose of this
study was to investigate the expression and the roles of TIMP-1 secreted
by Epstein-Barr-virus (EBV)-immortalized B lymphocytes. TIMP-1 was
isolated from conditioned medium of interleukin (IL)-1β stimulated
EBV-B lymphocytes; purified TIMP-1 was identified by mass spectrometry and
immunochemistry. TIMP-1-free MMP-9 was quantified after purification by
zymography and enzyme-linked immunosorbent assay. EBV-B
lymphocyte-secreted **TIMP**-1 **inhibited MMP**-9
gelatinolytic activity resulting in decreased B-cell transmigration as
measured in vitro. The release of huge amounts of TIMP-1 in proportion to
MMP-9 from B lymphocytes after EBV transformation was shown to be
correlated with secretion of IL-10 and dependent on culture time. In

contrast, there was little TIMP-1 and almost no IL-10 released from native B cells, suggesting a possible IL-10 mediated autocrine regulation mechanism of TIMP-1 synthesis. The MMP-9/TIMP-1 imbalance observed in the culture medium of EBV-B lymphocytes (TIMP-1>MMP-9) and of native B cells (MMP-9>TIMP-1) is suggestive of a new function for TIMP-1. We propose that TIMP-1 acts as a survival factor controlling B-cell growth and apoptosis through an autocrine regulation process involving IL-10 secreted by EBV-B lymphocytes. (C) **2000** Elsevier Science B.V.

L5 ANSWER 13 OF 15 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1999:196812 CAPLUS
DN 131:97052
TI Inhibition of Endothelial Cell Migration by Gene Transfer of Tissue Inhibitor of Metalloproteinases-1
AU Fernandez, Harold A.; Kallenbach, Klaus; Seghezzi, Graziano; Grossi, Eugene; Colvin, Stephen; Schneider, Robert; Mignatti, Paolo; Galloway, Aubrey
CS Department of Surgery, NYU Medical Center, Section of Cardiothoracic Surgery, New York, NY, 10016, USA
SO Journal of Surgical Research (1999), 82(2), 156-162
CODEN: JSGRA2; ISSN: 0022-4804
PB Academic Press
DT Journal
LA English
AB Angiogenesis requires degradation of the vessel's basal lamina and endothelial cell migration into the tissue stroma. Matrix metalloproteinases (MMPs) and their tissue inhibitors (TIMPs) play important roles in this process. MMP activity is tightly regulated during vessel growth. This work was designed to characterize the effect of TIMP-1 upregulation on endothelial cell invasion of the extracellular matrix. We constructed replication-deficient recombinant adenoviruses that encode either TIMP-1 (Ad.TIMP-1) or Escherichia coli lac Z (Ad. βgal) cDNA. Bovine aortic endothelial (BAE) cells were infected with 100 infectious particles/cell.

Gene expression was assessed by Northern and Western blotting. TIMP-1 activity in cell-conditioned media was measured by a resorufin-labeled casein protease assay. BAE cell migration was measured by Boyden chamber assays with 0.2% gelatin-coated, 8.0-μm polycarbonate membranes.Results. TIMP-1 was overexpressed by Ad.TIMP-1-infected BAE cells relative to control, Ad. βgal-infected or uninfected cells. TIMP-1 activity in Ad.TIMP-1 cell-conditioned medium was 2.8-fold higher than in control cells. By Boyden chamber assays with gelatin-coated membranes, Ad.TIMP-1-infected BAE cells showed 89.97 ±1.64% (mean ± SEM) reduction in migration relative to Ad.βgal-infected cells ($P < 0.02$) and 90.53 ± 1.12% relative to uninfected cells ($P < 0.02$). Without gelatin coating, migration was equivalent in all groups. The replication-deficient recombinant adenovirus we constructed affords rapid and efficient upregulation of functional TIMP-1 in endothelial cells. Infection results in a dramatic decrease in cell migration and invasion of extracellular matrix. Thus, such a recombinant vector may provide a useful tool for the gene therapy of vascular remodeling and inhibition of angiogenesis. (c) **1999** Academic Press.

RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 14 OF 15 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1998:521650 CAPLUS
DN 129:275230
TI Short chain fatty acids inhibit human (SW1116) colon cancer cell invasion by reducing urokinase plasminogen activator activity and stimulating TIMP-1 and TIMP-2 activities, rather than via MMP modulation
AU Emenaker, Nancy J.; Basson, Marc D.
CS Department of Surgery, Yale University School of Medicine, New Haven, CT, 06520-8062, USA
SO Journal of Surgical Research (1998), 76(1), 41-46
CODEN: JSGRA2; ISSN: 0022-4804
PB Academic Press
DT Journal

LA English
AB Short-chain fatty acids (SCFA) derived from dietary fiber may protect against invasive colon cancer by modulating degradative matrix metalloproteinases (MMPs) and protective tissue inhibitor of matrix metalloproteinases (TIMPs). Since the invasion depends on the MMP /**TIMP** ratio, SCFA may **inhibit** the colon cancer invasion by **inhibiting MMPs** and stimulating **TIMPs**. SW1116 colon cancer cells were seeded onto Matrigel-coated Boyden chambers and treated with unsupplemented media or media containing 10 mM acetate, propionate, or butyrate. SW1116 invasion was quantitated by light microscopy and conditioned media were assayed by ELISA for MMP-1, 2, 3, and 9, TIMP-1 and 2, MMP/TIMP complex, and urokinase plasminogen activator (uPA). Although all 3 SCFA inhibited the invasion, butyrate was more potent than acetate or propionate. Butyrate inhibited the SW1116 invasion by 35±1% of control vs. 18±9% for acetate and 10±6% for propionate. MMP-2 was not modulated by any of the 3 SCFA, while MMP-1 was modulated only by butyrate and MMP-3 by propionate. Acetate did not modulate MMPs, TIMP-1, or uPA, but stimulated TIMP-2. Propionate and butyrate stimulated MMP-9 and TIMP-2 by 119-233% and both inhibited uPA by 8-16%. TIMP-1 was stimulated only by butyrate and inhibited by propionate. Only butyrate stimulated both TIMP-1 and TIMP-2. Thus, dietary fiber may protect against invasive colon cancer through stimulation of **TIMP** and **inhibition** of uPA activities, rather than through SCFA effects on the activities of the MMPs studied.

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 15 OF 15 MEDLINE on STN DUPLICATE 5
AN 97080953 MEDLINE
DN PubMed ID: 8922288
TI Progelatinase B forms from human neutrophils. complex formation of monomer/lipocalin with TIMP-1.
AU Kolkenbrock H; Hecker-Kia A; Orgel D; Kinawi A; Ulbrich N
CS Deutsches Rheumaforschungszentrum Berlin, Germany.

SO Biological chemistry, (1996 Jul-Aug) 377 (7-8) 529-33.
Journal code: 9700112. ISSN: 1431-6730.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199702
ED Entered STN: 19970305
Last Updated on STN: 20000303
Entered Medline: 19970218
AB The three forms of neutrophil gelatinase B-monomer, homodimer and monomer/lipocalin complex-, were isolated from phorbolester stimulated neutrophil granulocytes by chromatography on gelatin-Sepharose and heparin-Ultrogel. On average, about 50% of the monomer/lipocalin complex was found to be complexed with TIMP-1. After activation with trypsin monomer, homodimer and monomer/lipocalin complex displayed a specific activity of about **2000** mU/mg towards the substrate N-(2,4)-dinitrophenyl-Pro-Gln-Gly-lle-Ala-Gly-Gln-D-Arg, whereas the monomer/lipocalin/TIMP-1 complex could be activated to a specific activity of only 200 mU/mg. The ternary monomer/lipocalin/TIMP-1 complex behaves like the progelatinase A-TIMP-2 complex and the progelatinase B-TIMP-1 complex in that it is an inhibitor for active metalloproteinases (MMPs) and, after activation, a gelatinase with a pronouncedly reduced activity. When the monomer/lipocalin/**TIMP**-1 complex **inhibits** an **MMP**, a quaternary complex monomer/lipocalin/TIMP-1/MMP is generated which after activation shows a sixfold higher proteolytic activity than the active ternary complex.